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## Response to 'Angiotensin-converting enzyme 2 (ACE2) gene and protein expression in diabetic patients without nephropathy'

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We thank Dr Amao<sup>1</sup> for the interest in our study of ACE2 expression in kidney biopsies of human subjects with diabetic nephropathy (DN), and other forms of glomerular disease.<sup>2</sup> Indeed, we did not study ACE2 gene or protein expression in biopsies of diabetic subjects without DN and, as mentioned in our discussion, it is not known whether the same observation would occur in such individuals. The feasibility of this type of study is questionable, given that it would be difficult to justify kidney biopsy in human subjects without clinical evidence of renal disease. Similarly, it is not known whether ACE2 expression in diabetic patients could be used to predict the development of DN. We also agree that the reported differences in ACE2 expression in different animal models of DN and human studies could relate to disease stage. Appreciation of the complexity of the renin-angiotensin system and its intrinsic balances continues to grow. We agree with Dr Amao that the potential influence of direct renin inhibition on the protective effects of ACE2 merits further study.

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2. Reich HN, Oudit GY, Penninger JM et al. Decreased glomerular and tubular expression of ACE2 in patients with type 2 diabetes and kidney disease. *Kidney Int* 2008; **74**: 1610–1616.

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## High-volume peritoneal dialysis in acute kidney injury

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**To the Editor:** In a study comparing high-volume peritoneal dialysis (HVPD) and daily hemodialysis (DHD), Gabriel et al.<sup>1</sup> report their experience from a randomized controlled trial. The authors report a mortality of 58% for HVPD and 53% for DHD. Subsequently, the article mentions 24 survivors in HVPD and 29 survivors in DHD, each group comprising 60 patients. Taking the figures of survivors into account, the mortality for the HVPD group should have been reported as 60% and that for the DHD group as 51.66% (or as 52%, the nearest whole number).

The study also compares the efficacy of HVPD in metabolic control with that of DHD. However, they excluded patients with severe hypercatabolism according to Schrier's criteria<sup>2</sup> at the time of randomization, giving the impression that the authors accepted the limitations of HVPD beforehand. Chitalia et al.<sup>3</sup> have earlier reported peritoneal dialysis to be reasonably effective in mild and moderate hypercatabolic acute renal failure. It would have been interesting to observe the efficacy of HVPD in severely hypercatabolic patients, especially when the authors have used very high volumes of 36–44 l/day of dialysis fluid for 7 days a week with a target Kt/V of 0.65/day.

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2. Schrier RW. Acute renal failure. *Kidney Int* 1979; **15**: 205–216.
3. Chitalia V, Almeida AF, Rai H et al. Is peritoneal dialysis adequate for hypercatabolic acute renal failure in developing countries? *Kidney Int* 2002; **61**: 747–757.

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## Response to 'Hormone therapy and loss of kidney function'

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We appreciate the interest of Dr Palya and colleagues<sup>1</sup> in our paper.<sup>2</sup> We agree that further exploration of the relationship between progestin use, either with or without estrogen, and loss of kidney function would be of great interest. The importance of the type of progestin contained in hormonal preparations has been previously highlighted.<sup>3,4</sup> Studies have suggested a link between adverse cardiac, vascular, and thrombotic events depending on the type of progestin exposure,<sup>3,4</sup> and indicate that the type of progestin in a hormonal preparation may play a role in